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Pain thresholds, pain-induced frontal alpha activity and pain-related evoked potentials are associated with antisocial behavior and aggressiveness in athletes



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ABSTRACT

Objectives: Deficiencies in perceptual and cognitive functions have been linked with antisocial and aggressive behavior. To test whether these putative relationships generalize to sport – a context where such behavior is common – we determined the extent to which pain thresholds and cortical activity in response to painful electrical stimulation were associated with antisocial and aggressive behavior in sport; we also examined their link to moral disengagement.

Design: A cross-sectional design was used.

Method: Ninety-four participants completed questionnaires, had their pain threshold determined, and then had their central and frontal pain-related cortical activity recorded while they were electrically stimulated at supra-threshold intensity.

Results: Subjective pain thresholds were positively related while pain induced frontal alpha power was negatively related to antisocial behavior and aggressiveness. Central pain evoked potential amplitudes were negatively related to aggressiveness and moral disengagement.

Conclusions: Sensitivity to and cortical processing of noxious stimuli were reduced in individuals who more frequently behave antisocially and aggressively when playing sport and who are more likely to use psychosocial maneuvers to justify their harmful behavior. Our findings reveal that pain-related deficits are a feature of individuals who engage in more frequent antisocial and aggressive behavior in the context of sport.

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1. Introduction

Sport is a social context where moral issues are highly relevant (for reviews see Kavussanu, 2008, 2012). Research has shown that during competitive games, team sport players deliberately foul, physically intimidate, and try to injure their opponents (e.g., Kavussanu, Seal, & Phillips, 2006; Kavussanu, Stamp, Slade, & Ring, 2009). Thus, it is important to improve understanding the factors associated with antisocial and aggressive behavior, which encompasses acts intended to harm or disadvantage another individual (Kavussanu, 2012) and harm another individual (Anderson & Bushman, 2002), respectively. Although much research has examined antisocial and aggressive behavior in sport from a social psychological perspective, more recently researchers have begun to

investigate this important topic from a *cognitive neuroscience* perspective (e.g., Kavussanu, Willoughby, & Ring, 2012; Micai, Kavussanu, & Ring, 2015). Research in non-sport contexts has revealed differences in how the brains of antisocial and aggressive individuals respond to sensory and cognitive demands compared to other individuals (for reviews see Blair, 2001; Volavka, 1990, 1999). For instance, these reviews discuss evidence that violent individuals are characterized by structural and functional abnormalities in their frontal and temporal lobes. We aimed to extend these findings to the sport context. In team sports that involve physical contact between players, such as association football, basketball, field hockey, and rugby, antisocial and aggressive behaviors are relatively common occurrences during games (Bredemeier & Shields, 1986; Kavussanu, 2012). Accordingly, the current study determined whether abnormal cortical processing and perception of pain is a feature of individuals who engage more frequently in antisocial and aggressive behavior when playing competitive team sport.

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1.1. Pain sensitivity

Antisocial behavior and emotional detachment are the two key defining features of psychopathy (Blair, 2001). Early clinical observations noted that psychopaths often fail to avoid punishment (Cleckley, 1959; Hetherington & Klinger, 1964). Experimental research has since documented that psychopaths are characterized by impaired aversive conditioning (Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002; Hare & Quinn, 1971; Lykken, 1957), blunted conditioned anticipatory arousal prior to an impending noxious stimulus (Hare, 1965), reduced blink responses to noxious stimuli (Benning, Patrick, & Iacono, 2005; Patrick, Bradley, & Lang, 1993), and reduced pain sensitivity (Fedora & Reddon, 1993; Hare, 1968; Hare & Thorvaldson, 1970; Schalling, 1971; Schalling & Levander, 1964). Taken together, these data suggest that the increased frequency of antisocial behavior in psychopaths may be linked to their relative insensitivity to aversive stimuli.

Further support for this proposal comes from studies showing that pain sensitivity is lower in aggressive and violent individuals (Niel, Hunnicut-Ferguson, Reidy, Martinez, & Zeichner, 2007; Reidy, Dimmick, MacDonald, & Zeichner, 2009; Seguin, Pihl, Boulerice, Tremblay, & Harden, 1996). Seguin et al. (1996) reported that boys with higher pain tolerance to pressure stimulation were characterized by increased history of physical aggression based on teacher reports. Niel et al. (2007) used the response choice aggression paradigm and found that males with higher pain tolerance to electrical stimulation administered higher intensity shocks and more maximal intensity shocks to their opponents. Similarly, Reidy et al. (2009) found that male (but not female) participants with higher pain tolerances scored higher on self-reported measures of verbal and physical aggression. Although the mechanism underlying this pain-aggression phenomenon has yet to be identified, a number of candidates have been mooted. It has been suggested that pain tolerant individuals may underestimate the degree of pain inflicted on their victims or may have been toughened up by frequent fights (Seguin et al., 1996). Based on this evidence, we tested the possibility that relative insensitivity to pain may be a feature of athletes who engage more frequently in antisocial and aggressive behavior when playing sport. In team contact sports, physical contact during competitive games can lead to unpleasant sensory and emotional experiences associated with tissue damage (i.e. pain). Antisocial behavior and aggression in team contact sports might be linked with pain sensitivity for various reasons: Pain tolerant athletes may be more likely to commit physical antisocial and aggressive acts because they cannot empathize with their victims (Stanger, Kavussanu, & Ring, 2012; Stanger, Kavussanu, Willoughby, & Ring, 2012) because of impaired cognitive perspective taking or emotional empathic concern and personal distress (cf. Seguin et al., 1996).

1.2. Pain-related evoked potentials

Researchers (e.g., Bromm & Lorenz, 1998) often supplement subjective reports of pain with its objective neurophysiological correlates to paint a more complete picture of the psychological and physiological processes implicated in the perception and processing of noxious stimuli. However, to our knowledge, no study has assessed cortical evoked potentials to painful stimuli to explore the central processes underlying the antisocial behavior–pain relationship. The electroencephalogram (EEG) represents a means of assessing cortical activity that involves the recording of electrical activity on the scalp to detect voltages

generated inside the brain. Evoked potentials represent the cortical activity elicited in response to the presentation of an exteroceptive stimulus, such as a painful electrical stimulus. The most commonly studied pain-related evoked potentials are the N2 and P2 potentials, which refer to the second negative and positive peaks, respectively, of the cortical response to a noxious stimulus and represent the cortical activity that results from processing a painful stimulus (Treede, Kenshalo, Gracely, & Jones, 1999). These scalp potentials are measured at the vertex because they are reliably found to be largest in amplitude at this location. Pain-related evoked potentials reflect pain processing, that is increasingly painful stimuli elicit increasingly larger potentials (Bromm & Lorenz, 1998). It is possible that attenuated pain-related evoked potentials are associated with the tendency to commit antisocial and aggressive acts.

1.3. Frontal cortical activity

There is evidence to suggest that frontal dysfunction, assessed using EEG, is a feature of aggressive individuals (Volavka, 1990). For instance, one study noted that violent behavior in psychiatric patients was negatively correlated with frontal alpha band EEG activity, particularly resting activity in the left hemisphere (Convit, Czobor, & Volavka, 1991). Similarly, brain imaging studies have implicated reduced prefrontal cortical activity (Raine, Buchsbaum, & LaCasse, 1997) and frontal lesions (Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994; Grafman et al., 1996) with antisocial and aggressive behavior. These observations are compatible with the proposal that aggressive behavior is determined by a circuit in the brain comprising the orbitofrontal cortex, anterior cingulate, and amygdala (Davidson, Putnam, & Larson, 2000). In EEG studies, prefrontal cortical activity is typically indexed by the amount of activity in the alpha frequency band: A fast Fourier transform is applied to the raw EEG waveform to yield the spectral power of the EEG signal with a frequency of between 8 and 12 cycles per second. High alpha activity was originally interpreted as cortical idling (Pfurtscheller, Stancak, & Neuper, 1996), but more recently has been viewed as reflecting a sensory gating mechanism involving inhibition of task-irrelevant and activation of task-relevant areas (Jensen & Mazaheri, 2010; Schürmann & Basar, 2001). Although it is possible to assess frontal alpha brain activity under resting conditions, recent research has found better results using stimulus induced activity (e.g., Coan, Allen, & McKnight, 2006). Taken together, there is sufficient evidence to suggest that relatively attenuated pain-induced frontal brain activity may be associated with the tendency to behave antisocially in sport.

1.4. Moral disengagement

Moral disengagement refers to the psychosocial mechanisms people use to minimize negative affect when they engage in transgressive behavior (Bandura, 1991; Boardley & Kavussanu, 2011). It allows individuals to engage in conduct that violates their personal standards without experiencing intense negative emotions that usually accompany such behavior. Moral disengagement operates by mentally reconstruing harmful behaviors into benign acts, minimizing personal accountability for harmful behavior, misrepresenting the injurious effects that result from such behavior, and blaming the nature or actions of the victim. Previous research has found that players who have the propensity to morally disengage are more likely to report engaging in antisocial behaviors toward other players (Boardley & Kavussanu, 2011). Given the link between blunted emotion and antisocial

behavior in violent offenders and psychopaths (e.g., Blair, 2001; Cleckley, 1959), it is possible that moral disengagement may be associated with attenuated sensitivity and responses to painful stimulation.

We could speculate on how moral disengagement might be linked with reduced pain. The distortion of consequences mechanism operates on the consequences of detrimental behavior and downplays the harm caused to victims: Individuals who minimize the harm they cause are more likely to repeat such actions (Bandura, 1999). Accordingly, athletes who feel little pain when they are hit, kicked or punched may also underestimate the seriousness of the injuries they cause (Boardley & Kavussanu, 2011), and, therefore are more likely to act aggressively (Niel et al., 2007; Reidy et al., 2009; Seguin et al., 1996). Some moral disengagement mechanisms operate on the agency of action by obscuring or minimizing one's role in the harm one causes (Bandura, 1999); these are displacement and diffusion of responsibility. It is possible that players who feel less pain are also those who obscure and minimize harm to others. With these possibilities in mind, the current study investigated the link between moral disengagement and pain.

1.5. The present study

In sum, research has highlighted a relationship between pain and antisocial/aggressive behavior in non-athletes. However, to our knowledge, no study has examined the links between pain sensitivity or cortical processing of painful stimuli and antisocial behavior, aggressiveness, and moral disengagement in sport. We aimed to extend previous research by obtaining both self-reported and cortical measures of pain to examine whether pain is related to antisocial behavior and aggressiveness (i.e., the tendency to become aggressive, Maxwell & Moores, 2007) in sport.

The first purpose of the study was to determine whether subjective pain thresholds, pain induced frontal alpha activity, and pain-related evoked potentials are associated with antisocial behavior and aggressiveness in sport. We expected that more frequent antisocial behavior and greater aggressiveness would be negatively associated with the subjective experience (i.e., higher pain thresholds) and cortical processing (i.e., less pain-induced frontal alpha activity, smaller pain-related evoked potential amplitudes) of pain. A second purpose was to determine whether pain (measured by subjective and objective methods) is related to moral disengagement in sport. We expected that moral disengagement would be negatively associated with self-reported pain and cortical measures of pain-related processing. Our hypotheses were tested using new analyses performed on an existing dataset (Kavussanu, Willoughby, & Ring, 2012).

2. Method

2.1. Participants

Ninety-four team sport athletes (48 males, 46 females), with a mean age of 20.95 ($SD = 2.72$) years and 8.51 ($SD = 4.31$) years playing experience were paid £20 for participating. Their main team sport was association football (31%), field hockey (26%), basketball (22%) rugby (18%), and water polo (3%). All sports were contact sports. We recruited athletes from these sports because contact sports have inherent potential for injury and therefore higher likelihood for antisocial and aggressive behavior to occur and moral issues to arise (Bredemeier & Shields, 1986). Athletes

from a variety of contact sports were recruited to increase the generalizability of our findings. All participants were free from neurologic and psychiatric disorders and medications. They were asked to refrain from alcohol, caffeine and smoking for at least 12 h prior to testing.

2.2. Self-report measures

2.2.1. Antisocial behavior

Antisocial behavior in sport was measured using the 8-item antisocial behavior toward opponents scale of the Prosocial and Antisocial Behavior in Sport Scale (Kavussanu & Boardley, 2009; Kavussanu, Stanger, & Boardley, 2013). Participants were asked to rate how often they engaged in different behaviors when playing their team sport. An example item is "Tried to injure an opponent". Each item was rated on a 5-point Likert scale, anchored by 1 (never) and 5 (very often). Kavussanu and Boardley (2009) reported very good internal consistency ($\alpha = .86$) for this subscale.

2.2.2. Aggressiveness

Competitive aggressiveness was measured using the 6-item aggressiveness scale of the Competitive Aggressiveness and Anger scale (Maxwell & Moores, 2007). The stem "When playing your team sport how often have you behaved, felt or thought that ..." was followed by six items measuring aggressiveness. An example item is "Violent behavior directed toward an opponent is acceptable". Each item was rated on a 5-point Likert scale, anchored by 1 (never) and 5 (very often). Maxwell and Moores (2007) have provided evidence for the factorial validity and reliability ($\alpha = .84$) of this scale.

2.2.3. Moral disengagement

Moral disengagement in sport was measured using the 8-item Moral Disengagement in Sport Scale—short (Boardley & Kavussanu, 2008). Participants were asked to indicate their level of agreement with a range of statements concerning thoughts and feelings they may have in sport on a 7-point Likert scale, anchored by 1 (strongly disagree) and 7 (strongly agree). This scale includes an item for each of the eight mechanisms of moral disengagement. An example item is "It is okay to treat badly an opponent who behaves like an animal". Boardley and Kavussanu (2008) reported very good internal consistency ($\alpha = .85$) for the scale.

2.3. Noxious stimulus

A noxious electrical stimulus was delivered using a nociception-specific concentric electrode designed to selectively activate A-delta fibers (Katsarava et al., 2006; Kaube, Katsarava, Kaufer, Diener, & Ellrich, 2000). Each stimulus consisted of a double pulse. Each rectangular wave pulse lasted 500 μ s separated by 100 μ s; this delay is below the threshold required to discriminate the two stimuli, thus they were perceived as a single stimulus. The stimulating electrode was placed on the supraorbital nerve above the left eye (Cuzalina & Holmes, 2005), and a constant current stimulator (Model DS7A, Digitimer Ltd, UK) provided the stimulation.

2.4. Pain threshold

The subjective pain threshold was determined using a two-stage procedure. First, the participant rated the electrical stimulation on a 5-point scale adapted from Tursky and O'Connell (1972): 0 (feel no sensation), 1 (feel any sensation), 2 (uncomfortable sensation), 3 (painful sensation), 4 (don't want to go any higher). The

stimulation was increased from .2 mA in steps of .2 mA until the stimulation became painful, that is the participant reported “3” on the scale. Using this as an initial estimate, the participant's pain threshold was determined using an up–down staircase procedure: Stimulus intensity was increased by .1 mA if the prior stimulation was rated as not painful by the participant, or decreased by .1 mA if it was rated as painful (Levitt, 1971). When the participant had reported that the stimulation was painful on three non-consecutive occasions, the pain threshold was calculated as the average of the last two peak stimulation values. These procedures have been used in previous research (cf., Wilkinson, McIntyre, & Edwards, 2013).

2.5. Pain induced alpha power and pain related evoked potentials

A noxious electrical stimulus, at an intensity corresponding to 125% of pain threshold ($M = 1.68$, $SD = 1.08$ mA), which was perceived as a single pinprick-like pain, was delivered while participants viewed a small black fixation cross on a white screen on six trials. On other trials (data not reported here), it was delivered while participants viewed a picture. The pictures were neutral (e.g., players standing or moving), pleasant (e.g., players celebrating, semi-naked players), and unpleasant (e.g., players being hurt or badly injured) in valence (for further details see Stanger, Kavussanu, Willoughby, et al., 2012). Electrical stimulation occurred every 20–25 s. The EEG was recorded using a 32-channel BioSemi ActiveTwo system (BioSemi, Netherlands), at 512 Hz, and was re-referenced to average earlobe electrodes offline. Electrophysiological data processing was performed using EEGLAB (Delorme & Makeig, 2004). The EEG was high-pass filtered using a finite impulse response windowed-sinc filter with a half-amplitude cut-off at 1 Hz and a .4 Hz transition band. We performed a fast Fourier transform (1 Hz bins) on the artifact-free epochs, and then computed power (dB) in the alpha (8–12 Hz) frequency band at left and right frontal (F3 and F4) sites in the seconds before and during the two second window following onset of painful stimulation. These values were then log-transformed and averaged across sites.

The key components of the pain-related evoked potential (see Fig. 1) are the amplitudes (measured in microvolts) of the second negative (N2) and positive (P2) peaks in the event related potential following painful stimulation (e.g., Edwards, Inui, Ring, Wang, & Kakigi, 2008). Thus, we calculated the N2 (in the 100–200 ms post-stimulation window) and P2 (in the 200–300 ms post-stimulation window) peaks. We focused on the Cz electrode, since this is where the N2 and P2 potentials are maximal (e.g., Katsarava et al., 2006). This was confirmed in the present study by examination of the scalp maps (see Appendix 1). The amplitude of the pain evoked potentials at the vertex (i.e., Cz) were measured relative to a 100 ms pre-stimulus baseline and calculated as the average amplitude of the seven data points around the peak value during the 100–200 ms time window for the N2 potential and the 200–300 ms time window for the P2 potential (i.e., the peak value and the three data points either side, corresponding to a window of approximately 12 ms).

2.6. Procedure

The study protocol was approved by the local research ethics committee and each volunteer gave informed consent to participate. At the start of the testing session, participants completed the self-reported measures of antisocial behavior, aggressiveness and moral disengagement. Following instrumentation, their pain

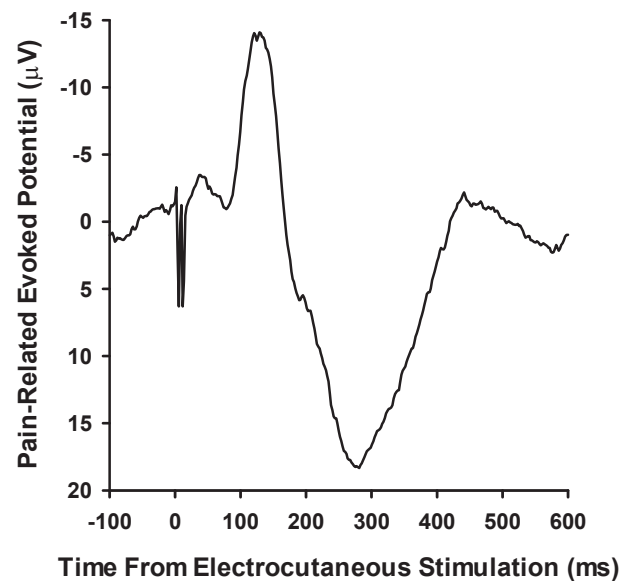


Fig. 1. Grand average evoked potential waveform recorded at Cz and elicited by noxious electrocutaneous stimulation of the supraorbital nerve. The N2 peak occurs 125 ms following stimulation whereas the P2 trough occurs 280 ms following stimulation.

threshold was determined. After sitting quietly for five minutes, they were instructed about the next task: They were told to always focus on the screen located in front of them and that their forehead would be stimulated when either a fixation cross or picture was on screen. In the task, the participant's pain related evoked potentials and pain induced frontal alpha activity were recorded as described above.

3. Results

3.1. Descriptive statistics and alpha coefficients

The means and standard deviations of the scales that measured antisocial behavior, aggressiveness, and moral disengagement are presented in Table 1. This table also presents alpha coefficients of the variables used in this study; internal consistency of all scales was good. On average, players reported that, when playing sport, they behaved antisocially toward opponents rarely or sometimes and reported aggressiveness rarely. They also reported moderate levels of moral disengagement. These scores are in line with those reported in previous research (e.g., Boardley & Kavussanu, 2008; Kavussanu & Boardley, 2009; Stanger, Kavussanu, Boardley, & Ring, 2013; Stanger, Kavussanu, & Ring, 2012; Stanger, Kavussanu, Willoughby, et al., 2012). Pearson correlations showed that moral disengagement was positively related to antisocial behavior, $r(92) = .48$, $p < .001$, and aggressiveness, $r(92) = .67$, $p < .001$.

The means and standard deviations of the subjective pain threshold, pain induced frontal alpha power, and pain evoked potential amplitudes are also shown in Table 1. The pain threshold for the noxious trigeminal stimulus is compatible with prior research (e.g., Katsarava et al., 2006). The scalp map for the pain related evoked potential confirmed that the N2 and P2 pain evoked potentials were maximal at the central electrode site Cz (see Appendix 1). We conducted a series of one-sample t-tests to determine whether the pain related evoked potentials and pain

Table 1
Descriptive statistics, Cronbach alpha coefficients and Pearson correlations.

Self-report measure	<i>M</i>	<i>SD</i>	α	Pain-related measures			
				Pain threshold (mA)	Frontal alpha power (dB)	Central N2 potential (μ V)	Central P2 potential (μ V)
Antisocial behavior (1–5)	2.37	.61	.79	.24*	–.22*	.07	–.07
Aggressiveness (1–5)	2.10	.61	.79	.20*	–.28**	.05	–.20*
Moral disengagement (1–7)	3.13	.94	.76	.10	–.15	.23*	–.27**
<i>M</i>				1.35	1.72	–18.09	23.95
<i>SD</i>				.87	2.91	–10.91	11.95

Note: * $p < .05$, ** $p < .01$.

induced alpha activity were significantly different from zero. These tests confirmed significant N2, $t(93) = 16.07$, $p < .001$, and P2, $t(93) = 19.43$, $p < .001$, pain evoked potentials (see Fig. 1). A one-sample t -test also confirmed that the pain induced frontal alpha activity was greater than zero, $t(93) = 5.74$, $p < .001$. Frontal alpha was lower in the seconds after noxious stimulation compared to the seconds before stimulation, $t(93) = 5.04$, $p < .001$, with the decrease averaging $-.96$ ($SD = 1.85$) dB.

3.2. Correlation analysis

Pearson correlations were computed between the pain variables (pain threshold, pain-induced frontal alpha, pain-related evoked potentials) and antisocial behavior, aggressiveness, and moral disengagement (see Table 1). The pain threshold was positively related to both antisocial behavior and aggressiveness: Players who acted more antisocially when playing team sport and players who reported more aggressiveness in sport tended to be less sensitive to noxious trigeminal stimulation. The pain threshold was not significantly related to moral disengagement. Frontal alpha power associated with the processing of noxious electrical stimulation was negatively associated with antisocial behavior and aggressiveness. These findings indicate that the players who acted more antisocially when playing team sports and players who displayed more aggressiveness in sport were characterized by less frontal alpha activity when exposed to painful stimuli. The negative N2 potential was positively related to moral disengagement, whereas the positive P2 potential was negatively related to moral disengagement and aggressiveness. In brief, smaller pain related evoked potential amplitudes were a feature of players who reported higher moral disengagement and aggressiveness in sport.

3.3. Gender as a moderator

Reidy et al. (2009) reported that the pain–aggression relationship was moderated by gender, with the effect evident for males but not females. To investigate this possibility in our study, we conducted moderation analysis using bootstrapping (Preacher & Hayes, 2008) and PROCESS for SPSS Release 2.13 (Hayes, 2013) to examine whether gender moderated the relationships between the pain variables (pain threshold, pain-induced frontal alpha, pain-related evoked potentials) and antisocial behavior, aggressiveness, and moral disengagement.

Bootstrapping was set at 5000 samples with bias corrected 95% confidence intervals; an effect was significant when the Confidence Interval (CI) did not contain zero. Results of these analyses indicated that the associations were not moderated by gender, $ts = .03$ – 1.45 , $ps = .15$ – $.97$, with one exception. Gender moderated the relationship between moral disengagement and the N2

component of the pain-related evoked potential, $b = 5.566$, 95% CI = .237, 10.895; $t = 2.08$, $p = .04$. Moral disengagement was associated with reduced N2 potential in males, $b = 4.113$, 95% CI = .790, 7.436; $t = 2.46$, $p = .02$, but not females, $b = -1.453$, 95% CI = -5.619 , 2.713; $t = .69$, $p = .49$.

4. Discussion

Deficiencies in pain processing have been linked with antisocial and aggressive behavior in diverse populations in non-sport contexts. To test whether these findings generalize to sport, we examined the extent to which pain thresholds and cortical activity in response to painful electrical stimulation were associated with antisocial behavior, aggressiveness and moral disengagement in the context of sport. Athletes' subjective pain threshold was positively related while pain induced frontal alpha power was negatively related to both antisocial behavior and aggressiveness. Moreover, central pain evoked potential amplitudes were negatively related to aggressiveness and moral disengagement. Thus, sensitivity to and cortical processing of noxious stimuli were reduced in both male and female athletes who behaved more antisocially, displayed more aggressiveness, and were more prone to morally disengage when playing competitive sport.

4.1. Pain sensitivity

In support of our hypothesis, relative insensitivity to painful electrical stimulation was a feature of athletes who engaged more frequently in antisocial conduct and who were more accepting of and willing to be aggressive when playing sport. This finding is compatible with the available literature in other contexts documenting reduced pain sensitivity in aggressive and violent individuals (e.g., Niel et al., 2007; Reidy et al., 2009; Seguin et al., 1996). In contrast to Reidy et al. (2009) gender did not moderate the pain–aggressiveness relationship. To date, no mechanism has been identified to account for these findings. Reduced pain sensitivity could lead to misattributions related to pain inflicted on a victim, whereby perpetrators who are less sensitive to and more tolerant of pain may misperceive the degree of pain experienced by another and, therefore, may be more willing to use violence during interpersonal conflict (Niel et al., 2007). Thus, athletes could engage in antisocial acts because they do not believe they are hurting their opponents as much as they really are. Our findings resonate with Niel et al.'s (2007) conclusion that insensitivity to pain (high pain tolerance) may increase the likelihood of aggression during an interaction where competition and provocation occur. Provocation occurs in competitive team sport, and therefore, aggressive competitors may not appreciate the consequences of their actions for

their opponents when playing sport. The current data provide preliminary evidence to support the proposal that relative insensitivity to pain is related to the frequency with which players engage in antisocial behavior when playing sport because regular perpetrators may underestimate the amount of pain inflicted on their victims.

4.2. Pain-related evoked potentials

We found that the N2 and P2 pain related evoked potentials were blunted in athletes reporting high levels of moral disengagement, while the P2 component of this potential was also blunted in athletes reporting high acceptance of aggression and willingness to be aggressive in sport. Moral disengagement mechanisms allow players to engage in transgressive conduct without experiencing strong negative emotions, such as guilt. We found that players who used more moral disengagement were more likely to report engaging in antisocial behaviors toward other players, in line with past studies (e.g., Kavussanu, Ring, & Kavanagh, 2015; Stanger et al., 2013; Stanger, Kavussanu, & Ring, 2012). Since the amplitudes of pain evoked potentials reflect pain processing, the current findings suggest that players who are more aggressive and use psychosocial maneuvers to justify their harmful behavior are more likely to have cortical deficits in how they respond to painful stimulation. Extending the previous behavioral research linking pain insensitivity to antisocial and aggressive behavior (e.g., Fedora & Reddon, 1993; Hare, 1968; Hare & Thorvaldson, 1970; Niel et al., 2007; Reidy et al., 2009; Schalling, 1971; Schalling & Levander, 1964; Seguin et al., 1996), the current study indicated that relatively attenuated pain evoked potentials are associated with the tendency to accept and excuse aggressive acts when playing sport.

4.3. Pain-induced frontal cortical activity

In line with our prediction, lower pain induced frontal brain activity was linked with increased frequency of antisocial behavior and greater acceptance of and willingness to aggress in sport. This finding is the first to link changes in frontal alpha to morally-relevant behavior in sport and is compatible with previous evidence showing that frontal dysfunction, assessed using EEG, is a feature of aggressive individuals (for review, see Volavka, 1990). In line with previous research (see Peng, Babiloni, Yanhui, & Hu, 2015), frontal alpha was suppressed in response to acute noxious stimulation, presumably reflecting the effects of a pain-related gating mechanism on frontal areas (Jensen & Mazaheri, 2010). Thus, antisocial behavior and aggressiveness were related to relatively low alpha oscillatory activity in the context of suppressed activity in the frontal regions of both hemispheres (cf. Convit et al., 1991). These results are also broadly compatible with brain imaging studies that have found a link between antisocial and aggressive behavior and prefrontal cortical activity (Raine et al., 1997) and frontal lesions (Damasio et al., 1994; Grafman et al., 1996); they are also in line with the suggestion that aggressive behavior is determined by a circuit in the brain comprising the orbitofrontal cortex, anterior cingulate, and amygdala (Davidson et al., 2000). In sum, by assessing pain induced frontal activation, we showed that reduced frontal alpha activation induced by noxious electrical stimulation is associated with the tendency to engage in harmful conduct when playing sport.

4.4. Study limitations and future research directions

Our study yielded some novel findings but also has some limitations which should be noted when interpreting the findings. First, only one stimulation site and one mode of stimulation were used, leaving open the possibility that different effects may be a feature of painful stimulation at other sites and other forms of painful stimulation. Accordingly, the generalizability of the observed effects from trigeminal nociception to other aspects of nociceptive processing remains to be confirmed in future research. Second, we employed a cross-sectional design and therefore causal relationships cannot be determined. Research using longitudinal and intervention designs that incorporate direct observations of behavior during sport participation and other assessments sensorimotor processes are now required. Finally, the correlation coefficients displayed in Table 1 ranged from $r = .20$ to $.28$, which, based on Cohen's (1992) definitions, where $.10$ is small, $.30$ is medium, and $.50$ is large, can be considered medium-to-small effects. These effect sizes should be used when interpreting the extent of the link between pain and antisocial behavior/aggressiveness. Nonetheless, this study has several strengths, including the use of a validated nociception-specific concentric electrode designed to selectively activate nociceptive afferents, state of the art equipment for measuring electroencephalographic signals, and a large sample size.

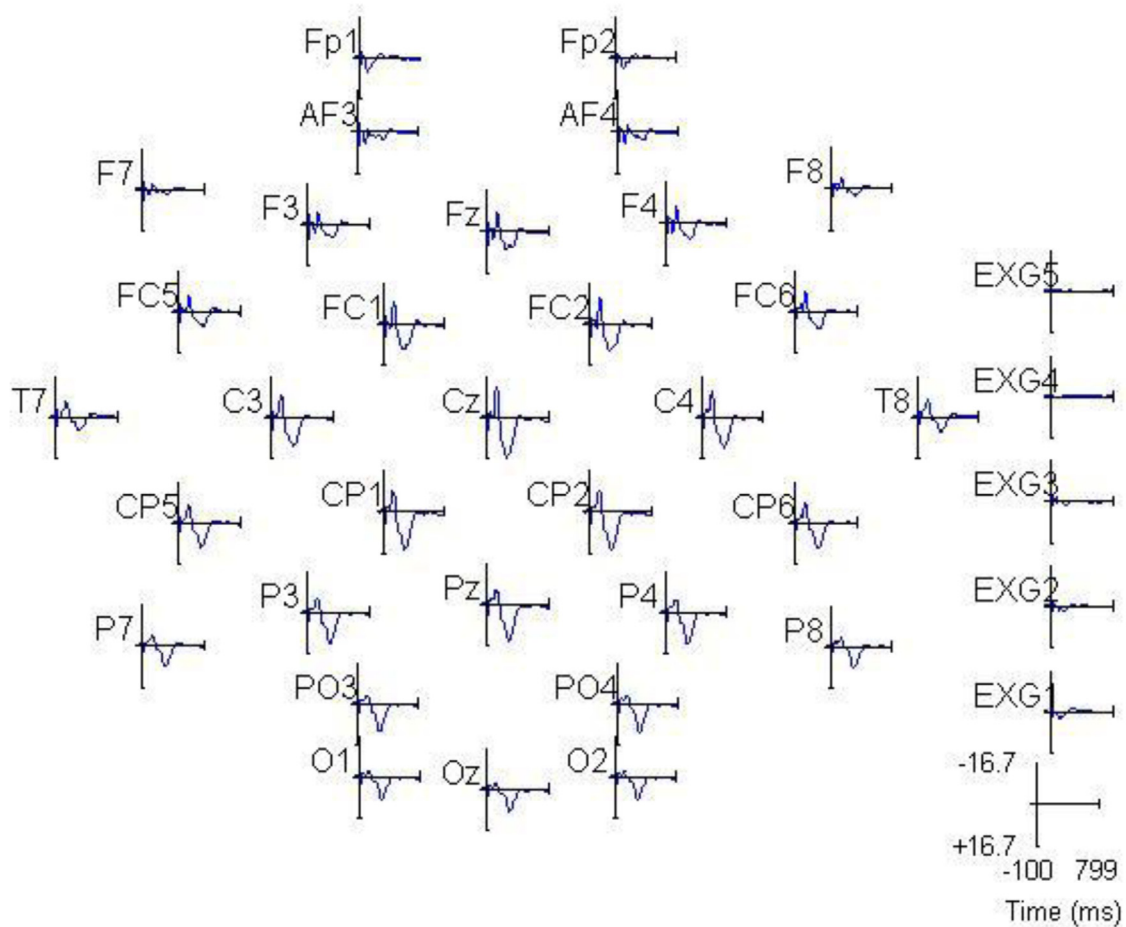
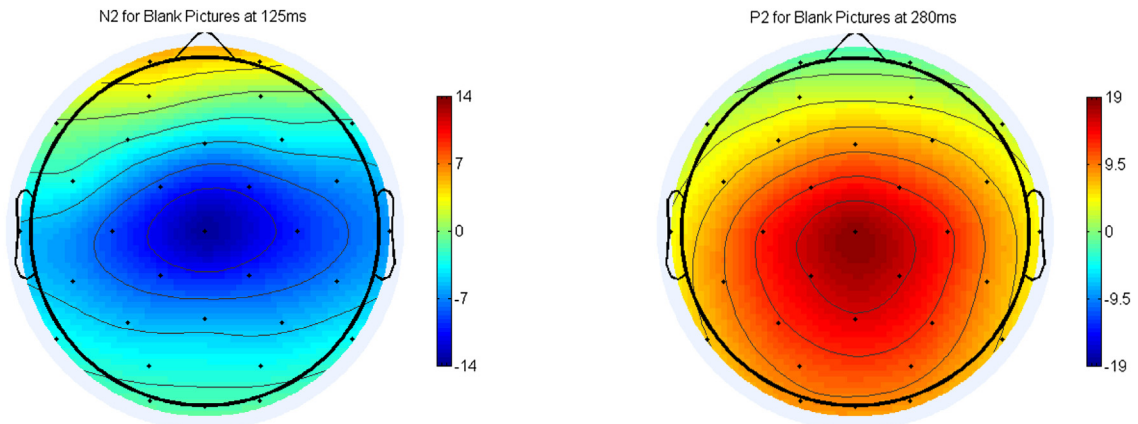
5. Conclusion

In conclusion, our findings revealed a consistent pattern indicating that players who perform and justify transgressive acts are characterized by relative insensitivity to noxious stimulation. Building on previous research showing that increased pain tolerance is associated with increased aggression (e.g., Niel et al., 2007; Reidy et al., 2009; Seguin et al., 1996), the current study is the first to confirm the pain–behavior relationship in objective neurophysiological indices of pain processing as well as a subjective measure of pain sensitivity. It may be worth noting that antisocial behavior seemed to be more closely associated with subjective pain threshold and alpha power whereas moral disengagement was more closely linked with cortical processing of pain. The reason for these differential relationships between moral variables and indices of pain sensitivity cannot be established from the present study. Accordingly, future research is now needed to establish the mechanisms underlying the pain–behavior and pain–cognition relationships. Our findings suggest that a profile comprising insensitivity to pain and blunted cortical responses to noxious stimulation may serve as a biobehavioral risk marker for antisocial and aggressive athletes. In sum, relative pain-related deficits are more likely to be a feature of individuals who engage more frequently in and excuse transgressive conduct in sport.

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Appendix 1. Top: Topographic scalp maps depicting the N2 and P2 peaks: The negativity was maximal at Cz at 125 ms post-stimulation while the positivity was maximal at Cz at 280 ms post-stimulation. Bottom: A scalp map showing the grand average evoked potential waveforms elicited by noxious electrocutaneous stimulation of the supraorbital nerve. Note that the N2–P2 complex was greatest at the vertex.



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